



UNIVERSITÀ
DEGLI STUDI
FIRENZE

FLORE

Repository istituzionale dell'Università degli Studi di Firenze

Pulse oximeter, the fifth vital sign: a safety belt or a prison of the mind?

Questa è la Versione finale referata (Post print/Accepted manuscript) della seguente pubblicazione:

Original Citation:

Pulse oximeter, the fifth vital sign: a safety belt or a prison of the mind? / Tozzetti C; Adembri C; Modesti PA.. - In: INTERNAL AND EMERGENCY MEDICINE. - ISSN 1970-9366. - STAMPA. - 4:(2009), pp. 331-332. [10.1007/s11739-009-0268-x]

Availability:

This version is available at: 2158/777211 since:

Published version:

DOI: 10.1007/s11739-009-0268-x

Terms of use:

Open Access

La pubblicazione è resa disponibile sotto le norme e i termini della licenza di deposito, secondo quanto stabilito dalla Policy per l'accesso aperto dell'Università degli Studi di Firenze (<https://www.sba.unifi.it/upload/policy-oa-2016-1.pdf>)

Publisher copyright claim:

(Article begins on next page)

Pulse oximeter, the fifth vital sign: a safety belt or a prison of the mind?

Camilla Tozzetti · Chiara Adembri ·
Pietro Amedeo Modesti

Received: 11 May 2009 / Accepted: 20 May 2009 / Published online: 13 June 2009
© SIMI 2009

Case report

Camilla Tozzetti

A 48-year-old woman hospitalized in the hematologic clinic because of chronic myeloid leukemia, during the night required the attention of the on-call physician for dyspnea associated with tachycardia. General physical examination revealed pallor and rhythmic tachycardia (145 beats/min). Her temperature was 98.6°F (37°C), respiratory rate was 32 breaths/min, blood pressure was 104/54 mmHg, and oxygen saturation was 95%. A complete blood count performed in the afternoon demonstrated an Hb of 9 g/dL, but the patient reported she had hematemesis in the late evening. “Did you give oxygen?”, asked the physician to the nurse. “No doctor, oxygen saturation is 95%. She called me for tachycardia, should I ask for the cardiologist?” Blood withdrawn was performed again, showing a further decrease in the Hb level (6 g/dL). Blood was requested and in the meantime colloid infusion started. The reluctance of the nurse was finally overcome and oxygen was added at 10 L/min. Tachycardia and dyspnea gradually relapsed.

Comment

Chiara Adembri, Pietro Amedeo Modesti

Oxygen saturation (SaO₂), together with measurements of heart rate, blood pressure, respiratory rate and temperature,

is now included among the essential vital sign assessment in patients care [1]. The routine monitoring of this parameter has increased the recognition of hypoxic episodes occurring in different clinical settings or during various procedures. However, while a low SaO₂ (<90–92%) always alarm care givers, there is also the risk of a false assumption, that is “normal” saturation (SaO₂ ≥ 94) means no hypoxia. When speaking with students, a misconception about the relationship between arterial oxygen partial pressure (PaO₂) and hemoglobin saturation is not rarely encountered [2] because oxygen delivered (DO₂) to tissue is perceived normal only on the basis of SaO₂ and PaO₂ [3]. Although the primary factor that determines how much oxygen is actually bound to hemoglobin is the partial pressure of oxygen (pO₂) in the hemoglobin solution, SaO₂ and PaO₂ cannot be considered alone without considering blood hemoglobin content nor cardiac output (CO) because DO₂ = CO × CaO₂ where CaO₂ is the oxygen content of blood. An anemic patient (Hb = 7 g/dL) may have an O₂ saturation of Hb of 100% with a pO₂ of 95 mmHg, thus looking deceptively non-hypoxic, although the total DO₂ of arterial blood is reduced by an half. With very low Hb values, DO₂ may even become inadequate to meet the tissue oxygen requirements (VO₂) for aerobic metabolism, a failure defining circulatory shock. Clinicians challenged to support DO₂ to avoid tissue hypoxia have to rapidly consider all the different components because CaO₂ is derived from hemoglobin content (Hb), SaO₂, and a constant K (the coefficient for hemoglobin–oxygen binding capacity) (CaO₂ = Hb × SaO₂ × K) [4]. Respiratory failure and inadequate DO₂ can thus result from malfunction of any aspect of the “ventilatory apparatus”, oxygen uptake in the lung via inspiration, passive oxygen diffusion into arterial blood, hemoglobin content and dissociation kinetics or from low CO. Each of these parameters

C. Tozzetti · C. Adembri · P. A. Modesti (✉)
Department of Critical Care Medicine and Surgery,
University of Florence, Viale Morgagni 85, Florence, Italy
e-mail: pamodesti@unifi.it

taken alone can fail to detect the presence of tissue hypoxia [4].

Marked dyspnea of BC, with the increased ventilatory rate and depth, was mainly due to the hypoxic stimulus of chemoreceptors. Carbon dioxide is the main stimulus to ventilation. However, an arterial PaO_2 of <60 mmHg also acts as an important stimulus. In particular carotid body senses soluble O_2 and signals the respiratory center to increase respiratory rate and depth (hypoxic ventilator response) [5]. Although O_2 solubility in blood is very low (0.003 mL/dL per mmHg, a constant independent of prevailing $p\text{O}_2$), O_2 content in plasma is a variable dependent on prevailing $p\text{O}_2$ (0.3 mL O_2 /dL). In conclusion, a FiO_2 increase cannot further enhance the oxygen Hb transport so that DO_2 cannot be significantly enhanced; however, soluble O_2 in plasma may increase thus reducing the stimulus at the chemoreceptors level. Oxygen administration might thus reduce the hypoxic ventilator response even if only

rapid plasma expansion with consequent CO increase may have increased DO_2 .

Conflict of interest statement The authors declare that they have no conflict of interest related to the publication of this manuscript.

References

1. Tansey EA (2008) Teaching the physiology of adaptation to hypoxic stress with the aid of a classic paper on high altitude by Houston and Riley. *Adv Physiol Educ* 32:11–17
2. Neff TA (1998) Routine pulse oximetry: a fifth vital sign? *Chest* 94:227
3. Cliff WH (2006) Case study analysis and the remediation of misconceptions about respiratory physiology. *Adv Physiol Educ* 30:215–223
4. Prakash ES, Madanmohan (2006) What does one mean by “arterial blood oxygenation?”. *Adv Physiol Educ* 30:46–47
5. Levy MM (2005) Pathophysiology of oxygen delivery in respiratory failure. *Chest* 128:547S–553S